ISSN: 0976-3031

Available Online at http://www.recentscientific.com

International Journal of Recent Scientific Research
Vol. 4, Issue, 10, pp.1631-1637, October, 2013

International Journal of Recent Scientific Research

RESEARCH ARTICLE

CHANGES IN HORMONAL ACTIVITIES OF THE FISH *CHANNA PUNCTATUS* (BLOCH) UNDER EXPOSURE TO DICOFOL 18.5 % (EC) SUB-LETHAL CONCENTRATION

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ARTICLE INFO

Article History:

Received 18th, September, 2013
Received in revised form 28th, September, 2013
Accepted 14th, October, 2013
Published online 28th October, 2013

Key words:
Dicofol, Hormones, LC_{50}, and *Channa punctatus*

ABSTRACT

Freshwater snake headed fish *Channa punctatus* (Bloch), was exposed to sub-lethal concentration ($1/10^{th}$ of 96 h LC_{50}) of Dicofol 18.5% EC for 24, 48, 72, 96 and 30 days and the consequential impact on hormonal activity was studied. The LC_{50} values calculated for 24, 48, 72, 96 h using Finney's (1972) probit analysis and were 0.075, 0.075, 0.72, and 0.72 ppm respectively. In the present study it was observed that, short-term dicofol exposure (96 h) did not exert appreciable changes in Estradiol and Testosterone levels in sub-lethal exposure. On the contrary, prolonged dicofol exposure (30Days) significantly decreased LH, FSH, TSH, T4, Testosterone and Progesterone levels in dose and time-dependent manner. The decrease in hormone action may be due to the interference of the test toxicant with the synthesis, transport, metabolism and elimination of hormones, thereby decreasing the concentration of natural hormones. The results obtained were in corroborative with the earlier reports and the discussed at length with the available literature.

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INTRODUCTION

A large number of chemicals occurring in our environment may have potential to interfere with the endocrine system of animals (Dalsenter, *et al.,* 1997). Many of these chemicals can disrupt development of the endocrine system and of the organs that respond to endocrine signals in organisms indirectly exposed during prenatal and/or early postnatal life; effects of exposure during development are permanent and irreversible (Colborn, *et al.,* 1993). Increasing interest has been observed among environmental and health institutions regarding the potential reproductive effects due to exposure to occupational and environmental chemicals (Dalsenter, *et al.*, 1997). Since the discovery of DDT in 1939 Mably *et al*., (1992), numerous pesticides (organochlorides, organophosphates, carbamates) have been developed and used extensively worldwide with few guidelines or restrictions. Some pesticides such as organochlorine, organophosphates and carbamates are known to cause morphological damage to the fish testis. These also affect female fish in the same way. They cause delayed Oocyte development and inhibition of steroid hormone synthesis, Kim (1998). Thyroid hormones play a key role in the maintenance of body homeostasis. Altered thyroid status may lead to changes in basal metabolic rate, lipid metabolism as well as cardiovascular, gastrointestinal and muscle function. Thyroid hormones are especially important during growth and development such as the maturation of the brain. A number of environmental agents can alter thyroid hormone levels in humans and animals. Hypothyroidism in rodents has been observed after exposure to PCB, TCDD, and chlorinated pesticides (Crisp *et al*., 1998). Disruption of the balance of endocrine hormones during development of young fish can also cause defects of the skeletal system, resulting in deformities and stunted growth Ewing, (1999); Goodbred,et al.,(1997). Survival and reproductive success of an organism requires an ability to adapt to constantly changing external environmental conditions, while maintaining a constant internal environment within very narrow tolerances (Goodman 2003). Some endocrine disruptors may exert their action by interfering with the brain's release of hormones, which in turn regulate the production of other hormones that control the growth and the activity of many other endocrine glands. Indeed, the pituitary has been termed the conductor of the endocrine orchestra, and pollutants that cause the pituitary region in the brain malfunction may therefore have multiple effects. In aquatic systems, OCPs easily join food webs, and their concentrations increase with each trophic level Sun *et al*. (2006). In the case of fish, accumulation of these compounds in gonads may result in reduced reproduction potential, as well as in a decrease in fry number and developmental disorders. Residues of organochlorine pesticides in edible parts of fish are another problem (Darko *et al*. 2008, Li *et al*., 2008). Among food products, fish are considered the main source of these compounds in human diet. Fish products sometimes contain significant amounts of these compounds that pose a health risk to consumers (Binelli and Provini 2004, Sun et al. 2006). As OCPs have the ability to induce endocrine, metabolic, and reproductive disorders, their concentrations in food products, especially in edible fish species, should be monitored. Fish have been also used in numerous studies as the most effective bioindicators in the environmental monitoring of aquatic ecosystems (Kannan *et al*., 1995, van der Oost *et al*., 2003, Hinck et al. 2008).

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MATERIALS AND METHODS

The test fish *Channa punctatus* size 10-12 $\pm$ ½ cm and weight 14-16 $\pm$ ½ gm were collected from local fish market and brought to the laboratory and stored in large plastic tubs containing tap water. The fish were acclimated to laboratory conditions at 28 $\pm$ 2^{o}C for 10 days and the water was well aerated daily, as per the recommendations of committee on toxicity tests of Aquatic organisms (A.P.H.A 1998). The fish were fed with groundnut oil cake and rice bran. The water in the fish storage tanks was changed daily. The feeding was stopped one day prior to experiments. Tap water was used for maintaining the fish in the fish tanks had a pH 7.2±0.1, dissolved oxygen 8.0±0.3 mg/L and bicarbonates 95.0±5.0mg/L at 28 $\pm$ 2^{o}C.

Hormone sample collection and storage

Serum: Using a serum separator tube (SST) the samples were collected and allowed the samples to clot for 30 minutes before centrifugation for 15 minutes at 1000 rpm. Serum was removed and assayed immediately or aliquot and stored samples at -20°C.

Plasma: Plasma was collected using citrate, EDTA, or heparin as an anticoagulant. Centrifuge for 15 minutes at 1000 rpm within 30 minutes of collection. Plasma GH levels were measured using a specific double-antibody Channa GH radioimmunoassay as outlined in Bjornsson *et al*., (1994). The GH assay requires duplicate 50-Al samples and there was insufficient plasma to measure plasma GH in parr in 1999. Plasma insulin-like growth factor I (IGF-I) and thyroxine (T4) were measured in duplicate 10 Al samples by radioimmunoassays as outlined in Moriyama *et al.* (1994).

RESULTS AND DISCUSSION

The calculated values for hormones and standard deviation, along with per cent change over the control in different hormones of fish *Channa punctatus* (Bloch), in different time periods graphs were given in Fig. A.1-6. The hormone levels of control fish were more or less stable during the 24 h, 48 h, 72 h, and 96 h respectively. Whereas at 30th day of the experiment all hormones levels in control were also decreased. This may be due to starved condition of the fish. The hormone levels decreased on exposure to sub-lethal concentration of Dicofol 18.5 % EC (Kelthane). They also showed a tendency of decrease with the increase in the period of exposure at 24h the decrement is about 5-20 % and by thirty days it reached to 30-60 %. The maximum level of change was observed in the hormone LH (74%) and minimum in Follicle stimulating hormone FSH (T4) (25%). Under exposure to sub-lethal Dicofol 18.5% EC, the hormone level was found to decrease in all above hormones of *Channa punctatus* (Bloch). Maximum decrease was observed at 30 days exposure to the toxicant.

In the present study it was observed that, short-term dicofol exposure (96 h) did not exert appreciable changes in Estradiol and Testosterone levels in sublethal dicofol 18.5% EC. On the contrary, prolonged dicofol-exposure (30Days) significantly decreased LH, FSH, TSH, T4, Testosterone and Progestirone levels in dose and time-dependent manner. In the case of estradiol and testosterone, a significant increase in the hormone level was observed. Many studies show a direct relationship between concentrations of pesticides and related chemicals in fish tissues and depressed hormone concentrations. Dicofol Inhibition of androgen synthesis, increase of estrogens synthesis, binding to estrogen receptor was reported by Thibaut and Porte (2004), Okubo.*et.al*. (2004). These results go hand in hand with those of, Tag El-Din, *et al*., (2003) who reported that dicofol at 4.19 and 16.75 mg/kg b.w./day, for 6 months, induced significant decrease in LH level in male rats. Pesticides are reported to cause degenerative changes in gonads and arrest gametogenic processes either by acting directly on the gonads or by interfering with the secretary activity of the hypothalamo-hypophyseal-gonadal/thyroid axis that regulates various reproductive events. Secretion of hormones such as gonadotropin-releasing hormone (GnRH), gonadotropin, growth hormone, adrenocorticotropic hormone (ACTH), testosterone, estrogens, 17,20β-dihydroxyprogesterone and thyroid hormones are in general lowered, leading to cessation of gametogenesis, vitellogenesis, Oocyte maturation, ovulation, spermiation, etc. Adverse effects of pesticides have also been demonstrated on fecundity, fertilization, hatching, and postembryonic development. The effects are highly variable and depend on the nature, dose, and mode of application of the pesticides. Lal, (2007). Changes in levels of various thyroid hormones during different reproductive phases occurred most probably due either to inhibit (in response to γ-BHC) or to stimulated (in response to malathion) extra thyroidal T_4 monodeiodination and their altered excretion or consumption rate after 4 weeks exposure of γ-BHC (8 and 16 ppm) and malathion (10 and 20 ppm),. Marked decline in LH and FSH levels in the present study confirm the findings of Desaulniers *et al*., (1999) and Lafuente *et al*., (2000), who investigated the toxicological influences of PCB (126 and 153) and methoxychlor, O.C, at different concentrations on male rats. On the other hand, the results of the present study disagree with the findings of Tag El-Din et al., (2003), who mentioned that FSH level increased significantly after treatment with dicofol at lower and higher-doses (4.19 and 16.75 mg/kg b. w./day), in drinking water, for 6 months. It was proved that certain O`C pesticides did not alter LH and FSH levels when administered into rats at different doses for short-term intervals, such as: DDT (Krause, 1977), PCB 28 and 77 (Desaulniers *et al.*, 1997) and TCPM (Foster *et al*., 1999).

Caroll *et al*., (1991) reported an elevation in circulating levels of inhibin, a glycoprotein of primarily sertoli cell origin which inhibits FSH synthesis and secretion by the pituitary could account for the observed decrease in serum FSH level which was confirmed histopathologically by degeneration and atrophy of seminiferous tubules including leydig and sertoli cells. FSH stimulates the sertoli cells of the seminiferous tubules to produce androgen binding protein, probably move via the sertoli cells to other germ cells and to the epididymus where the testosterone is released to exert its physiological effects in sperm maturation (Mably *et al.*, 1992), Saravanan. *et.al* (2010) reported that sub-lethal concentrations of endosulfan chronic exposure to *Labeo rohita* (50 days), caused increased levels of serum T3 (Triodothyronine) but TSH level decreased in treated fish. Singh and Singh (1992b) observed suppression of testosterone,estradiol and 17 a –Ohprogesterone in ovarian tissue and plasma (Singh and Singh, 1991), greater reduction in ovarian hormones at higher doses of pesticides and impaired steroidogenesis by inhibiting gonadotropin secretion (Singh and Singh, 1992a),

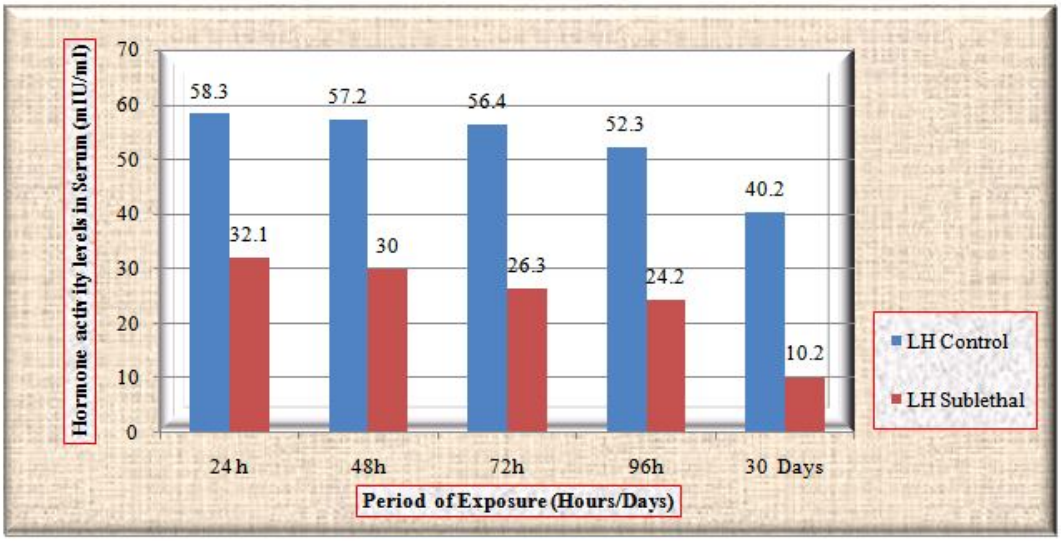

Fig 1: Changes in the total LH hormone activity levels (mIU/ml) and % change over the control in different hours/days (24h, 48h, 72h, 96h and 30days) of the fresh water fish *Channa punctatus* exposed to sub-lethal concentration of Dicofol 18.5% EC.

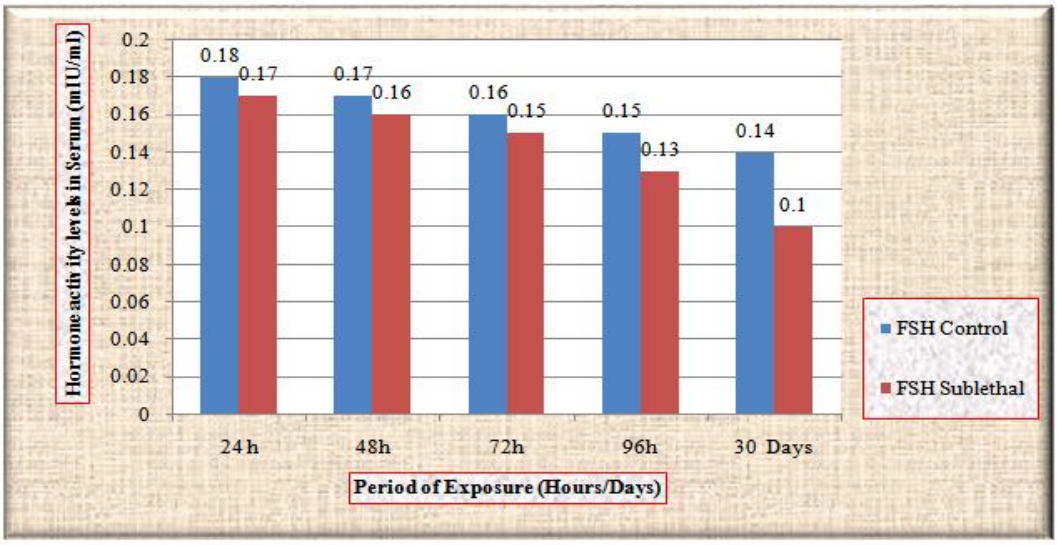

Fig. 2: Changes in the total FSH hormone activity levels (mIU/ml) and % change over the control in different hours/days (24h,48h,72h,96h and 30days) of the fresh water fish *Channa punctatus* exposed to sub-lethal concentration of Dicofol 18.5% EC.

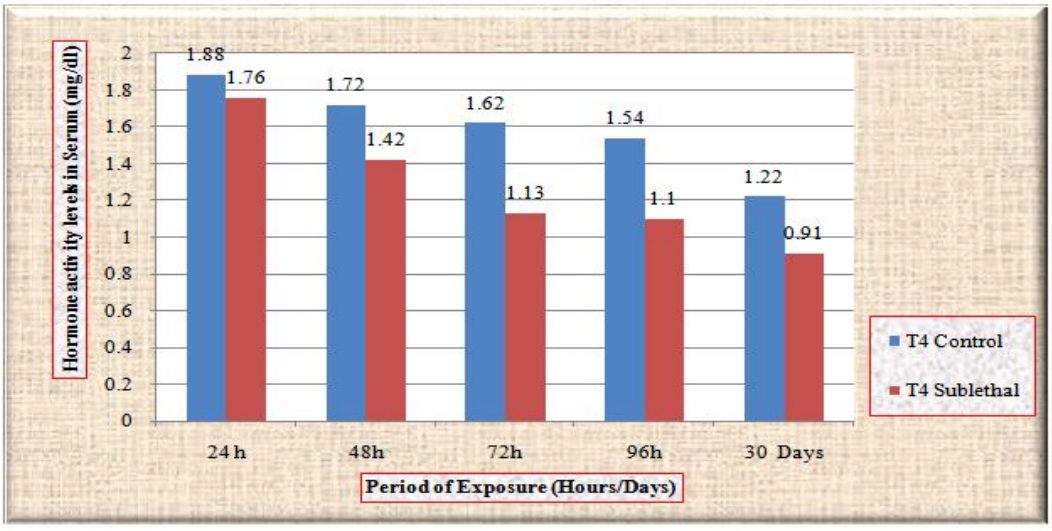

Fig. 3: Changes in the total T4 hormone activity levels (mg/dl) and % change over the control in different hours/days (24h,48h,72h,96h and 30days) of the fresh water fish *Channa punctatus* exposed to sub-lethal concentration of Dicofol 18.5% EC.

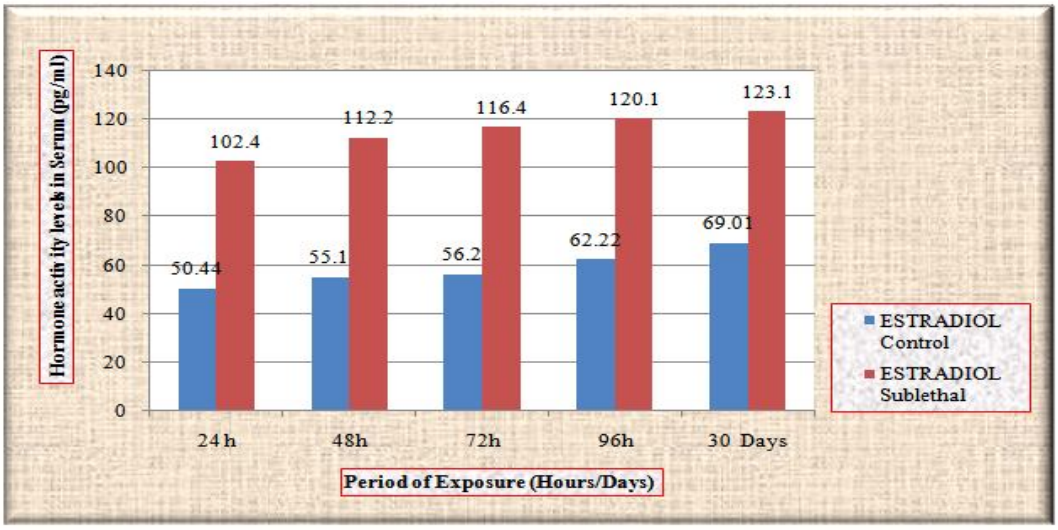

Fig. 4: Changes in the total Estradiol hormone activity levels (pg/ml) and % change over the control in different hours/days (24h,48h,72h,96h and 30days) of the fresh water fish *Channa punctatus*, exposed to sub-lethal concentration of Dicofol 18.5% EC.

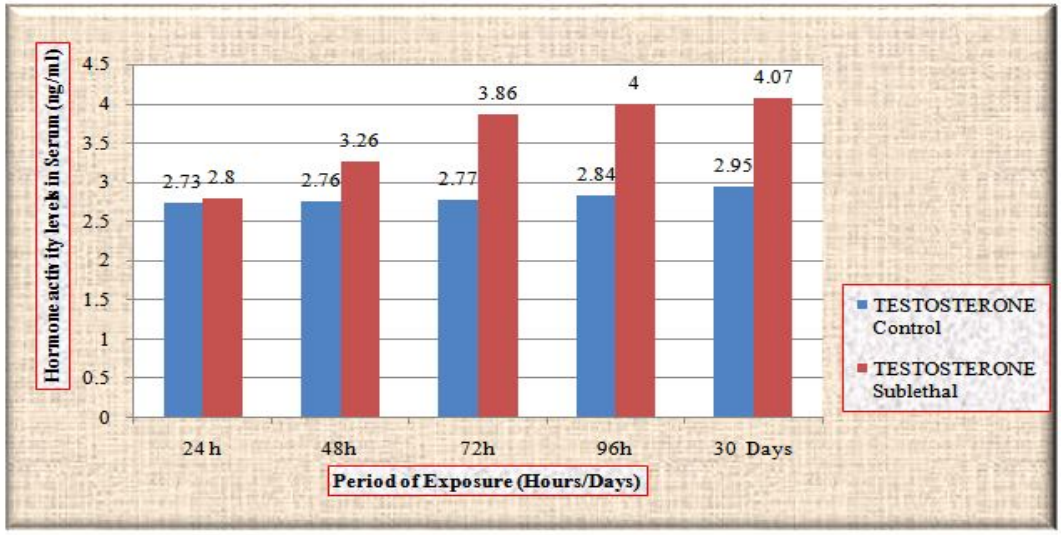

Fig. 5: Changes in the total Testosterone hormone activity levels (ng/ml) and % change over the control in different hours/days (24h, 48h, 72h, 96h and 30days) of the fresh water fish *Channa punctatus.* exposed to sub-lethal concentration of Dicofol 18.5% EC.

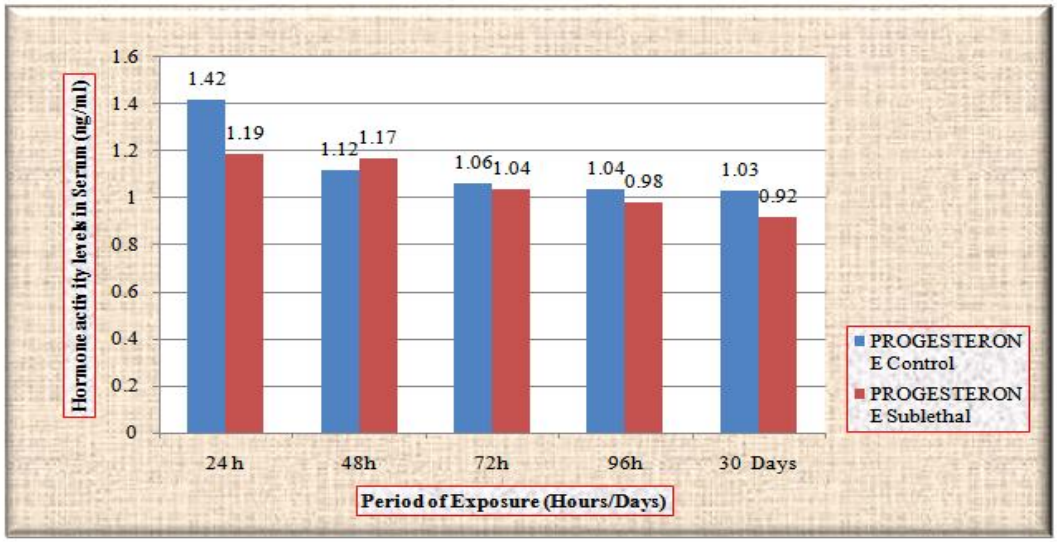

Fig. 6: Changes in the total Progesterone hormone activity levels (ng/ml) and % change over the control in different hours/days (24h, 48h, 72h, 96h and 30days) of the fresh water fish *Channa punctatus.* exposed to sub-lethal concentrations of Dicofol 18.5% EC.

decreased ovarian phospholipids, checking of hydrolysis of esterified cholesterol to free cholesterol, impairment of translocation of lipid to ovary by inhibiting hypothalamo-hypophysial-ovarian axis (Singh and Singh, 1992b), inhibition of de-esterification of esterified cholesterol to free cholesterol and steroidogenesis and hormone secretion into plasma (Singh *et al.,* 1993) have been reported in singhi catfish treated with varying doses of malathion and y-BHC over different periods. According to the suggestion of Singh and Pandey (1990), the changes in the pattern of the steroidogenic enzymes 3 ß-hydroxysteroid dehydrogenase and 17 ß-hydroxysteroid dehydrogenase lead to inhibition of testicular androgen biosynthesis in adult rats, which is required for spermatogenesis in seminiferous tubules and sperm maturation in the epididymus. A complementary proposed mechanism, could explain dicofol induced toxicity, is blocking gonadotropin production and/or release by the pituitary, thereby testosterone production by leydig cells is not stimulated, causing spermatogenesis arrest **(**Vanage, *et al.,* 1997). This mechanism is supported by the data previously reported by Mably *et al.,* (1992).

Reproductive hormones and vitellogenin may be suppressed in fish exposed to xenobiotic chemicals in the field or laboratory (Folmar, 1993). Endocrine disruption in freshwater fish presenting intersex individuals with Ovotestes has now been reported from many places and in many freshwater and marine fish species (Jobling *et al*., 1998). Indirectly, endocrine disruption might also affect fat storage due to specific chemicals, some of them mimicking the steroid hormone estrogen (Turner and Sharpe, 1997). The significant decrease of testosterone levels may be as a result of direct damage caused by dicofol on leydig cells, the main site of testicular androgen biosynthesis. Results of the present work agree with those reported by Krause (1977), Desaulniers *et al*., (1999), Lafuente *et al*., (2000), Ben Rhouma *et al.,* (2001) and Choudhary and Joshi (2003), who noted that T level was significantly decreased in male rats treated with organochlorine pesticides at different doses, i.e. DDT, PCB-126 and 153, methoxychlor, DDT, endosulfan, respectively. Similar results were recorded with O.C pesticides at different experimental period; i.e. DDT (Ben Rhouma *et al*., 2001), lindane (Chitra *et al.,* 2001 and Sujatha *et al.,* 2001) and endosulfan (Choudhary and Joshi, 2003). Brown and Casida (1987) and Jadaramkunti and Kaliwal (2002) showed that reduction of testes and epididymus weights in rats treated with the highest dose of dicofol for long-term are the result of reduction diameter of semniferous tubules, spermatogenic, Leydig and Sertoli cells.

Kim, D.E. (1998) stated that some pesticides such as organochlorine, organophosphates and carbamates are known to cause morphological damage to the fish testis in males and ovary in females and cause delayed Oocyte development and inhibition of steroid hormone synthesis. Maxwell and Dutta, (2005) in their study have correlated the changes in the ovarian follicles of bluegills exposed to diazinon and the estradiol levels in the blood, which led to the endocrine disruption. The teleost testis, as in mammals, is composed of steroid hormone-secreting endocrine interstitial cells and sperm-producing lobular or tubular compartments (Nagahama, 1983). The reproductive potential of fish is affected, when reared in water containing pesticide residues (Moore and Waring, 2001). The physiological functions of fish get altered upon exposure to different pesticide concentrations (Gupta and Saxena, 2006). Among the key events in the female reproductive process is ovulation, which is regulated by the endocrine and central nervous systems. Doses of pesticides that are not high enough to kill fish are associated with subtle changes in behavior and physiology that impair both survival and reproduction (Kegley S, *et al.,* 1999). In the preent study it was proved that the organ chlorine insecticide Dicofol 18.5% EC in sub-lethal concentration had induced changes in the activity levels of hormones of the fresh water snake headed fish *Channa punctatus.* The present findings were in agreement with the earlier findings of several authors. The present study emphasizes the need to enlighten the farmers about the adverse effects of indiscriminate use of pesticides on the crops.

Acknowledgements

The authors thank the Head of the Department of Zoology, Acharya Nagarjuna University for providing necessary facilities. They also thank UGC for providing equipment to carry out this work under Special Assistance Programme.

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